AJH 2005; 18:792–796

Alcohol Consumption and Augmentation Index in Healthy Young Men: The ARYA Study

Marijke J.C.A. van Trijp, Joline W.J. Beulens,
Willem J.W. Bos, Cuno S.P.M. Uiterwaal,
Diederick E. Grobbee, Henk F.J. Hendriks, and Michiel L. Bots

Background: Light-to-moderate alcohol consumption is associated with a decreased risk of ischemic cardiovascular disease, whereas increased alcohol intake is related to hypertension and intracerebral hemorrhage. We studied the effect of alcohol consumption on the augmentation index (AIx), a measure of arterial wave reflection in a population of healthy young men.

Methods: Three hundred twenty-nine men (mean age 28 years) from the Atherosclerosis Risk in Young Adults study (ARYA-study) were studied. The level of alcohol consumption and risk factors for cardiovascular disease were determined. The AIx was estimated by radial applanation tonometry using a Sphygmocor device. The relation between alcohol intake level and AIx was determined using linear regression models.

Results: There was a positive graded relation between alcohol intake and AIx. Subjects who did not drink, who drank 1 to 2 glasses/d, or who drank ≥3 glasses of alcohol/d had, respectively, a −0.6% (95% confidence interval [CI] −4.2, 3.0), 0.2% (95% CI −2.6, 2.9), and 3.4% (95% CI 0.2, 6.7) difference in AIx compared with very light drinkers (<1 glass/d). After adjustment for current smoking, body mass index and HDL-cholesterol, those consuming >3 glasses/d had a 3.29% (95% confidence interval CI 0.01, 6.7) higher AIx compared with those consuming <1 glass/d.

Conclusions: In a population of healthy young men, the heaviest drinkers had a significantly higher AIx. This finding supports the evidence that increased alcohol consumption is related to vascular damage at young age. Am J Hypertens 2005;18:792–796 © 2005 American Journal of Hypertension, Ltd.

Key Words: Augmentation index, alcohol, young adults.

Moderate alcohol consumption is associated with reduced ischemic cardiovascular disease risk compared to no alcohol consumption and excessive alcohol consumption. These studies support the protective concept of moderate alcohol consumption.[1] The reduced risk of cardiovascular disease (CVD) with moderate alcohol consumption may be explained by an increase of HDL cholesterol,[2] a decrease in inflammatory factors such as C-reactive protein,[3] or an increase in fibrinolytic activity.[4] In addition, several studies in the middle-aged and the elderly have indicated that moderate alcohol consumption may be associated with reduced carotid intima media thickness[5] and reduced aortic stiffness or pulse wave velocity (PWV).[6,7] Apart from effects of alcohol on vascular characteristics and cardiovascular events, increased alcohol has been shown to increase the risk of hypertension.[8,9] Data on the relation of alcohol with vascular structure come mostly from studies in middle-aged or elderly persons. Few data are available on the effect of excessive levels of alcohol consumption at young age and therefore we set out to study the vascular effects of alcohol in young adult men by measuring the augmentation index (AIx).

Augmentation index is a measure of arterial wave reflection, defined as the difference between early and late pressure peaks divided by pulse pressure,[10] and is proposed as a marker for CVD,[11] which may reflect "vascular damage" in its early stage. An increased AIx has been related to risk factors of CVD, such as elevated blood pressure (BP), hypercholesterolemia,[12] diabetes,[13] and smoking.[14] In addition, an increased AIx is associated with CVD risk[15,16] and with prevalent CVD.[11] Furthermore, because alcohol consumption

Received August 26, 2004. First decision December 1, 2004. Accepted December 4, 2004.

From the Julius Center for Health Sciences and Primary Care, University Medical Center Utrecht (MJCAvT, CSPMU, DEG, MLB), Utrecht; Department of Nutritional Physiology, TNO Nutrition and Food Research (JWJB, HFJH), Zeist; Department of Human Nutrition, Wageningen University (JWJB), Wageningen; and Department of Internal Medicine, St. Antonius Hospital (WJWB), Nieuwegein, The Netherlands.

Address correspondence and reprint requests to Dr. M.L. Bots, Julius Center for Health Sciences and Primary Care, University Medical Center Utrecht, Str. 6.131, P.O. Box 85500, Heidelberglaan 100, 3508 GA/ 3584 CX Utrecht, The Netherlands; e-mail: m.l.bots@umcutrecht.nl

0895-7061/05/$30.00
doi:10.1016/j.amjhyper.2004.12.011

© 2005 by the American Journal of Hypertension, Ltd.
Published by Elsevier Inc.

may have acute vasodilatory[17,18] and BP-lowering[19] effects, it may affect wave reflections. However, chronic alcohol consumption of more than 30 to 60 g/d is associated with an increased BP.[1,8,9] The effect of alcohol consumption on AIx has not been investigated frequently. Therefore, we investigated the effect of alcohol consumption on AIx in young, healthy, male adults.

Methods

Study Design and Population

The rationale and design of the Atherosclerosis Risk in Young Adults (ARYA) study have been described elsewhere.[20] In short, the Utrecht cohort of the ARYA study consists of 750 young adults (352 men, 398 women) born between 1970 and 1973, who attended secondary school in the city of Utrecht, the Netherlands, and of whom the original medical records from the Municipal Health Care were available. These records contained data about birth parameters such as birth weight, length at birth, and gestational data. To be eligible for the ARYA-study birth weight had to be known and also data of at least one BP measurement obtained at adolescence. From October 1999 to December 2000 the participants visited the outpatient clinic of our department twice in a 3-week period. The Medical Ethical Committee of the University Medical Center Utrecht approved the ARYA-study and all participants gave written informed consent. As the variation in alcohol consumption is much larger in men than in women, we decided to restrict the study to the male subjects of the cohort.

Cardiovascular Risk Profile at Young Adulthood

During the first visit, anthropometric measurements were performed. Height, weight, and waist-to-hip ratio were measured with indoor clothes without shoes. At each visit BP was measured twice after 5 min rest with an interval of 5 to 15 min on the left arm in a sitting position using a semiautomated device (Dynamap; Critikon, Tampa, FL) without replacing the cuff between the two measurements. Mean systolic and diastolic BP were calculated as the mean of two BP measurements. Pulse pressure (PP) was calculated as (systolic BP − diastolic BP) and mean arterial pressure (MAP) was calculated as (2 × diastolic BP + systolic BP)/3. Despite that the Dynamap provides MAP estimates directly, unfortunately during the study only the systolic and diastolic BP levels were entered into the ARYA database. A written standardized questionnaire was completed on cardiovascular risk factors, such as smoking pattern, alcohol consumption, and family history of (risk factors for) cardiovascular disease. In the ARYA questionnaire subjects had to select 1 of 5 options to categorize their usual alcohol intake. (0; <1; 1 to 2; 3 to 5, and >5 glasses/d). During the second visit fasting venous blood samples were drawn. Total cholesterol (TC), HDL cholesterol, triglycerides (TG), and glucose were determined using a Vitros950 dry-chemistry analyzer (Johnson & Johnson, Rochester, NY). Low-density lipoprotein cholesterol was calculated using the Friedewald formula. Carotid–femoral PWV was measured as described previously.[21]

Measurement of AIx

Vascular measurements were performed using the SphygmoCor Blood Pressure Analysis System (Sydney, Australia). Aortic pulse waveform, AIx, and central aortic pressures were derived by aplanation tonometry on the radial artery. Radial pressure waveforms were recorded with a micromanometer (Millar SPT-301, Millar Instruments, Houston, TX). The obtained pressure waveforms were calibrated with a peripheral BP value of the brachial artery. Subsequently, the radial pressure waveforms were transformed into a single calibrated waveform. Ascending aortic pressure was derived from the central pressure waveform, using a generalized transfer function that is incorporated in the SphygmoCor device.[22] Finally, AIx (the difference between early and late pressure peaks divided by pulse pressure) was calculated and expressed as a percentage.

Measurements were repeated in a subset of 28 subjects in a period of several weeks to assess the reproducibility of the AIx measurement. The intraclass correlation coefficient of the repeated measurements was 0.65, unadjusted for BP differences between the visits.

Data Analysis

For a few subjects data on alcohol consumption, laboratory tests, or AIx were missing ($n = 23$). Therefore, data of 329 subjects were available for the analyses. Because the group that consumed more than 5 glasses/d was small ($n = 7$), it was pooled with the group of subjects that consumed 3 to 5 glasses/d.

The association of alcohol consumption with AIx was examined in two ways. First, AIx was calculated for each alcohol intake level adjusted for age, height, heart rate, and MAP using general linear models. These adjustments were based on the literature indicating that these factors are major determinants of Aix.[23–26] To validate the self-reported information on alcohol intake we studied the relation of alcohol intake level with HDL-cholesterol level as increased alcohol intake is related to an increase in HDL-cholesterol.[2] Second, multivariable regression analysis using dummy variables was performed to investigate relation of alcohol intake level with AIx. For this analysis the group drinking less than 1 glass/d was used as a reference ($n = 141$). After adjustment for age, height, heart rate, and MAP (model A), current smoking and BMI were added to the model (model B). The HDL-cholesterol was added to determine whether the alcohol effect was related to HDL-cholesterol level (model C). Finally, PWV was added to the model because it is positively related to AIx, as an increased PWV results in earlier wave reflec-

tions and thus a higher AIx. Furthermore, there is evidence that PWV is inversely related to alcohol consumption.[6,7]

The P values $< .05$ were regarded statistically significant. Data were analyzed using SPSS version 11.0 for Windows (SPSS, Chicago, IL).

Results

The general characteristics of the study population are presented in Table 1. Those factors that were related to alcohol intake showed linear relationships. The HDL-cholesterol level gradually increased with increasing alcohol intake. There was no evidence for nonlinear relationships between general characteristics and alcohol intake. Age, height, heart rate, and MAP adjusted AIx increased with increasing alcohol intake: from 2.4 (standard error 1.6), to 3.0 (0.9), to 3.2 (1.1), to 6.5 (1.4) with alcohol intake levels of 0, <1, 1 to 2, and 3 or more glasses/d. When BMI and current smoking were additionally included in the model (model B in Table 2), the group with a drinking level of ≥3 glasses/d remained to have a statistically significant higher AIx compared with the group drinking <1 glass/d. Further adjustment for HDL-cholesterol (model C) did not essentially change this result (Table 2).

The result of model A in Table 2 seemed to indicate that the shape of the relation was linear, whereas models B and C seemed to indicate a more J-shaped relation. We further explored this by fitting a model with an added quadratic component. This, however, did not improve the fit of the model (R^2 increased from 16.7% to 16.9%), and thus not supporting the concept of a J-shaped relation.

Because in earlier studies alcohol had been related to PWV ($n = 229$), we performed adjustment for PWV (model D, Table 2). This, however, did not affect the magnitude of the relation with AIx for the group with the highest drinking level in comparison with the reference group.

Discussion

This report from the ARYA-study shows that young subjects who drink ≥3 glasses alcohol per day have a significantly higher AIx compared to persons who drink <1 glass/d. This relation remained after adjustment for risk factors, HDL-cholesterol and PWV.

To appreciate the findings some limitations of the present study need to be discussed. First, for the assessment of alcohol intake a questionnaire was used. This might have introduced misclassification of exposure as selective underreporting might have occurred in the heavier drinking groups.[27] Our finding that increasing HDL-cholesterol is related to increasing alcohol consumption argues against a major misclassification. In addition, generally misclassification in alcohol intake will lead to an attenuation of the associations. Second, no information was available on the type of alcoholic beverage, changes in drinking behavior, and drinking pattern. The first issue does unlikely affect our results considerably, as most of the effect of alcohol is due to the alcohol itself rather than to other components in the drink.[28] Changes in drinking behavior will probably not be important, because our study population consists of young adults. Yet, because binge drinking occurs frequently in young male adults,[29] drinking pattern may have affected our results. Unfortunately it was not possible to address binge drinking because this information was not collected. Third, the AIx was estimated from the radial pressure waveform

Table 1. General characteristics of the study population ($n = 329$ men) by alcohol consumption categories

	0 glasses/day	<1 glass/day	1–2 glasses/day	≥3 glasses/day
Number of subjects	42	141	90	56
Age (y)	28.5 (0.98)	28.4 (0.89)	28.3 (0.95)	28.3 (0.85)
SBP (mm Hg)	133 (15)	133 (12)	131 (11)	132 (12)
DBP (mm Hg)	75.5 (11)	73.9 (7.9)	72.5 (8.4)	75.4 (9.8)
PP (mm Hg)	58.2 (10)	58.7 (10)	58.7 (8.5)	56.4 (10)
MAP (mm Hg)	95 (12)	94 (8)	92 (9)	94 (10)
Heart rate (beats/min)	66 (9)	63 (9)	61 (9)	61 (10)
Height (cm)	182 (7)	183 (6)	185 (6)	185 (7)
Weight (kg)	80.6 (12)	84.8 (14)	82.4 (12)	82.0 (12)
BMI (kg/m^2)	24.3 (3.8)	25.2 (3.6)	24.1 (3.4)	24.0 (3.1)
Waist/hip ratio	0.89 (0.07)	0.90 (0.06)	0.87 (0.06)	0.87 (0.06)
Glucose (mmol/L)	5.0 (0.4)	5.2 (1.6)	5.1 (0.4)	5.3 (0.8)
Triglycerides (mmol/L)	1.14 (0.64)	1.45 (0.75)	1.26 (0.63)	1.39 (0.27)
Total cholesterol (mmol/L)	4.6 (0.9)	4.9 (1.0)	4.7 (1.0)	4.8 (0.8)
LDL-cholesterol (mmol/L)	2.8 (0.93)	3.1 (0.90)	2.8 (0.96)	2.86 (0.77)
HDL-cholesterol (mmol/L)	1.30 (0.33)	1.23 (0.28)	1.34 (0.30)	1.39 (0.27)
Current smokers (%)	7.1	33.3	44.4	46.4
Ejection duration (ms)	319 (21)	326 (18)	330 (18)	332 (17)
Augmentation index	1.6 (11.5)	2.98 (10.7)	3.26 (10.9)	6.93 (11.7)
Pulse wave velocity (m/s)	6.44 (0.82)	6.25 (0.79)	6.40 (1.17)	6.20 (0.65)

Values are means with standard deviations in parentheses, or percentages.

using a generalized transfer function.[22] The generalized transfer function was developed with data of a study population of men and women with an indication for diagnostic cardiac catheterization and our study population differs in that respect. Studies have been performed comparing "true" invasively measured AIx and estimated AIx by applanation tonometry, indicating that the estimated AIx is sometimes underestimated[22,30] or overestimated[31] and the correlation between measured and estimated AIx varies from relatively good[30] to relatively poor.[31] Currently, use of the transfer function is debated.[32–35] The previous mentioned studies[22,30,31] were not clear as to whether the difference between the true measured values and the estimated values is a random phenomenon or whether it is due to a shift in the distribution. If the first would be true this would lead to an underestimation of the true associations (bias toward the null value). If the second would be the case associations would be valid, but the magnitude of the associations would be too high or too low. We assume that if the transfer function would generate complete random values we would find no associations at all, and this is not the case in our study.

To the best of our knowledge, this is the first study in a population of young healthy adults, showing that alcohol consumption of ≥3 units/d results in a significantly higher AIx. Mahmud and Feely[36] studied both acute and chronic effects of alcohol consumption and the effects on AIx. In a study among 3 men and 5 women (age range 21 to 40 years) a decrease in AIx was found 90 min after consumption of 500 mL of red wine, independent from the effect of alcohol on BP.[36] In the same article, they reported on findings of a study population of 324 men and women (age range 18 to 86 years). The AIx was higher (12% ± 2%) in men with excessive (>21 units/week, $n = 67$) alcohol intake than those with less (<21 units/week) alcohol intake (5% ± 1%). When differences in BP were taken into account, the statistical significance was lost. For women, no relation was found. Unfortunately,[36] no dose-dependent data was provided for the relation of alcohol consumption and AIx in a dose-dependent manner. Sierksma et al[6] studied the dose-dependent relation of alcohol consumption on wave reflections and PWV among healthy postmenopausal women aged 50 to 74 years. For PWV an inverse relation was shown, whereas for AIx no relation was observed when physiologic parameters as age, height, and ejection duration were taking into account. A similar report by Sierksma et al[7] but in middle-aged and elderly men showed a J-shaped relation of alcohol with PWV. In those middle-aged men, alcohol consumption was one of the determinants of AIx, showing a U-shaped relationship independent of physiologic factors (van Trijp MJCA, et al. unpublished) The observed differences between studies may be due to differences in validity of the assessment of alcohol consumption (between men and women), in sample size, in pathophysiology of the pathway of how alcohol may affect wave reflections, and in the prevalence of other risk factors related to AIx. In the latter case it may become more difficult for alcohol to show relations with AIx when other factors are more prevalent (eg, increased age).[37]

Results of our study have been adjusted for the most important determinants of AIx (ie, age, height, heart rate, and MAP). Additional adjustment for BMI and current smoking did not influence the relation of alcohol consumption with AIx. Addition of HDL-cholesterol to the model did not materially change the relation. Furthermore, adjustment for PWV did not change the relation, suggesting that the relation of alcohol with AIx is independent from arterial stiffness. However, the relation between PWV and AIx in this dataset was not very strong (Spearman correlation coefficient of 0.14) and given the moderate reproducibility of both PWV and AIx, adjustment may not completely remove the arterial stiffening component from the relation.

In our study subjects with the highest drinking level had a significantly higher AIx. Although an increased AIx has been related to elevated levels of cardiovascular risk factors,[11–16] the precise clinical implications of an increased AIx needs to be established.

In conclusion, this study provides evidence that in a cohort of young male adults, subjects drinking ≥3 or more alcoholic beverages per day have a significantly higher AIx in comparison with subjects drinking <1 beverage per day.

Table 2. Change in AIx (%) per alcohol intake level compared with very light drinkers

Alcohol Intake Level (glasses/d)	*N*	Model A	Model B	Model C	Model D
0	42	−0.61 [−4.19; 2.97]	0.34 [−3.30; 3.97]	0.35 [−3.30; 3.99]	−0.502 [−4.76; 3.72]
<1	141	Reference	Reference	Reference	Reference
1–2	90	0.15 [−2.60; 2.89]	0.01 [−2.74; 2.77]	0.03 [−2.75; 2.81]	0.11 [−3.27; 3.48]
≥3	56	3.44 [.23; 6.65]*	3.27 [.04; 6.50]*	3.29 [.01; 6.58]*	4.01 [.18; 8.08]*

Values are linear regression coefficients [95% confidence interval].
Model A: adjusted for age, height, heart rate, and MAP.
Model B: adjusted for age, height, heart rate, MAP, current smoking, BMI.
Model C: as model B but with HDL-cholesterol.
Model D: as model C but with PWV.
* $P < .05$.

Additional Reference

van Trijp MJCA, Bos WJW, Muller M, van der Schouw YT, Grobbee DE, Bots ML: Alcohol consumption and arterial wave reflection in middle aged and elderly men (submitted).

References

1. Grobbee DE, Rimm EB, Keil U, Renaud S: Health issues related to alcohol consumption, in (ed): Alcohol and the Cardiovascular System. Oxford, Blackwell Sciences Ltd., 1999, pp 125–179.
2. Rimm EB, Williams P, Fosher K, Criqui M, Stampfer MJ: Moderate alcohol intake and lower risk of coronary heart disease: meta-analysis of effects on lipids and haemostatic factors. BMJ 1999;319: 1523–1528.
3. Sierksma A, van der Gaag MS, Kluft C, Hendriks HF: Moderate alcohol consumption reduces plasma C-reactive protein and fibrinogen levels; a randomized, diet-controlled intervention study. Eur J Clin Nutr 2002;56:1130–1136.
4. Hendriks HF, Veenstra J, Velthuis-te Wierik EJ, Schaafsma G, Kluft C: Effect of moderate dose of alcohol with evening meal on fibrinolytic factors. BMJ 1994;308:1003–1006.
5. Mukamal KJ, Kronmal RA, Mittleman MA, O'Leary DH, Polak JF, Cushman M, Siscovick DS: Alcohol consumption and carotid atherosclerosis in older adults: the Cardiovascular Health Study. Arterioscler Thromb Vasc Biol 2003;23:2252–2259.
6. Sierksma A, Lebrun CE, van der Schouw YT, Grobbee DE, Lamberts SW, Hendriks HF, Bots ML: Alcohol consumption in relation to aortic stiffness and aortic wave reflections: a cross-sectional study in healthy postmenopausal women. Arterioscler Thromb Vasc Biol 2004;24:342–348.
7. Sierksma A, Muller M, van der Schouw YT, Grobbee DE, Hendriks HF, Bots ML: Alcohol consumption and arterial stiffness in men. J Hypertens 2004;22:357–362.
8. Witteman JC, Willett WC, Stampfer MJ, Colditz GA, Kok FJ, Sacks FM, Speizer FE, Rosner B, Hennekens CH: Relation of moderate alcohol consumption and risk of systemic hypertension in women. Am J Cardiol 1990;65:633–637.
9. Thadhani R, Camargo CA Jr, Stampfer MJ, Curhan GC, Willett WC, Rimm EB: Prospective study of moderate alcohol consumption and risk of hypertension in young women. Arch Intern Med 2002; 162:569–574.
10. Davies JI, Struthers AD: Pulse wave analysis and pulse wave velocity: a critical review of their strengths and weaknesses. J Hypertens 2003;21:463–472.
11. Weber T, Auer J, O'Rourke MF, Kvas E, Lassnig E, Berent R, Eber B: Arterial stiffness, wave reflections, and the risk of coronary artery disease. Circulation 2004;109:184–189.
12. Wilkinson IB, Prasad K, Hall IR, Thomas A, MacCallum H, Webb DJ, Frenneaux MP, Cockcroft JR: Increased central pulse pressure and augmentation index in subjects with hypercholesterolemia. J Am Coll Cardiol 2002;39:1005–1011.
13. Wilkinson IB, MacCallum H, Rooijmans DF, Murray GD, Cockcroft JR, McKnight JA, Webb DJ: Increased augmentation index and systolic stress in type 1 diabetes mellitus. QJM 2000;93:441–448.
14. Mahmud A, Feely J: Effect of smoking on arterial stiffness and pulse pressure amplification. Hypertension 2003;41:183–187.
15. Nurnberger J, Keflioglu-Scheiber A, Opazo Saez AM, Wenzel RR, Philipp T, Schafers RF: Augmentation index is associated with cardiovascular risk. J Hypertens 2002;20:2407–2414.
16. Duprez DA, Kaiser DR, Whitwam W, Finkelstein S, Belalcazar A, Patterson R, Glasser S, Cohn JN: Determinants of radial artery pulse wave analysis in asymptomatic individuals. Am J Hypertens 2004; 17:647–653.
17. Hashimoto M, Kim S, Eto M, Iijima K, Ako J, Yoshizumi M, Akishita M, Kondo K, Itakura H, Hosoda K, Toba K, Ouchi Y: Effect of acute intake of red wine on flow-mediated vasodilatation of the brachial artery. Am J Cardiol 2001;88:1457–1460.
18. Teragawa H, Fukuda Y, Matsuda K, Higashi Y, Yamagata T, Matsuura H, Chayama K: Effect of alcohol consumption on endothelial function in men with coronary artery disease. Atherosclerosis 2002;165:145–152.
19. Rosito GA, Fuchs FD, Duncan BB: Dose-dependent biphasic effect of ethanol on 24-h blood pressure in normotensive subjects. Am J Hypertens 1999;12:236–240.
20. Oren A, Vos LE, Uiterwaal CS, Bak AA, Gorissen WH, Grobbee DE, Bots ML: The Atherosclerosis Risk in Young Adults (ARYA) study: rationale and design. Eur J Epidemiol 2003;18:715–727.
21. Oren A, Vos LE, Uiterwaal CS, Grobbee DE, Bots ML: Aortic stiffness and carotid intima-media thickness: two independent markers of subclinical vascular damage in young adults? Eur J Clin Invest 2003;33:949–954.
22. Chen CH, Nevo E, Fetics B, Pak PH, Yin FC, Maughan WL, Kass DA: Estimation of central aortic pressure waveform by mathematical transformation of radial tonometry pressure. Validation of generalized transfer function. Circulation 1997;95:1827–1836.
23. Gatzka CD, Cameron JD, Dart AM, Berry KL, Kingwell BA, Dewar EM, Reid CM, Jennings GL: Correction of carotid augmentation index for heart rate in elderly essential hypertensives. ANBP2 Investigators. Australian Comparative Outcome Trial of Angiotensin-Converting Enzyme Inhibitor- and Diuretic-Based Treatment of Hypertension in the Elderly. Am J Hypertens 2001;14:573–577.
24. Kelly R, Hayward C, Avolio A, O'Rourke M: Noninvasive determination of age-related changes in the human arterial pulse. Circulation 1989;80:1652–1659.
25. Smulyan H, Marchais SJ, Pannier B, Guerin AP, Safar ME, London GM: Influence of body height on pulsatile arterial hemodynamic data. J Am Coll Cardiol 1998;31:1103–1109.
26. Wilkinson IB, MacCallum H, Hupperetz PC, van Thoor CJ, Cockcroft JR, Webb DJ: Changes in the derived central pressure waveform and pulse pressure in response to angiotensin II and noradrenaline in man. J Physiol 2001;530:541–550.
27. Feunekes GI, van't Veer P, van Staveren WA, Kok FJ: Alcohol intake assessment: the sober facts. Am J Epidemiol 1999;150:105–112.
28. Rimm EB, Klatsky A, Grobbee DE, Stampfer MJ: Review of moderate alcohol consumption and reduced risk of coronary heart disease: is the effect due to beer, wine, or spirits. BMJ 1996;312: 731–736.
29. Webb E, Ashton CH, Kelly P, Kamali F: Alcohol and drug use in UK university students. Lancet 1996;348:922–925.
30. Segers P, Qasem A, De Backer T, Carlier S, Verdonck P, Avolio A: Peripheral "oscillatory" compliance is associated with aortic augmentation index. Hypertension 2001;37:1434–1439.
31. Hope SA, Tay DB, Meredith IT, Cameron JD: Use of arterial transfer functions for the derivation of aortic waveform characteristics. J Hypertens 2003;21:1299–1305.
32. O'Rourke MF, Nichols WW: Use of arterial transfer function for the derivation of aortic waveform characteristics. J Hypertens 2003;21: 2195–2197.
33. Hope SA, Tay DB, Meredith IT, Cameron JD: Reply. J Hypertens 2003;21:2197–2199.
34. Avolio AP, O'Rourke MF: Applicability and benefit of arterial transfer functions. J Hypertens 2003;21:2199–2200.
35. Hoeks AP: Reply. J Hypertens 2003;21:2200–2201.
36. Mahmud A, Feely J: Divergent effect of acute and chronic alcohol on arterial stiffness. Am J Hypertens 2002;15:240–243.
37. Mitchell GF, Parise H, Benjamin EJ, Larson MG, Keyes MJ, Vita JA, Vasan RS, Levy D: Changes in arterial stiffness and wave reflection with advancing age in healthy men and women: the Framingham Heart Study. Hypertension 2004;43:1239–1245.